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Appellee's Brief 1975-SC-1072

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**KYSC1975-SC-1072-02**

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# **APPELLEE'S BRIEF**

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# SUPREME COURT OF KENTUCKY

File No. 75-1072

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ROSA BROACH, Individually and as Adminis-  
tratrix of the Estate of Sammy S. Broach,  
Deceased, and

WORKMEN'S COMPENSATION BOARD - Appellants

VERSUS

UNITED STATES STEEL CORPORATION;  
JAMES R. YOCOM, Commissioner of Labor and  
Custodian of the Special Fund - - Appellees

---

APPEAL FROM HARLAN CIRCUIT COURT  
HONORABLE JAMES C. BROCK, JUDGE

---

## BRIEF FOR APPELLEES

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FEB 27 1976

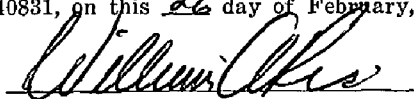
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Pursuant to RCA 1.250 true copies of this brief were mailed to Smith & Carter, P. O. Box 710, Harlan, Kentucky 40831; Honorable William L. Huffman, Director, Workmen's Compensation Board, Frankfort, Kentucky 40601; and to Honorable James C. Brock, Judge, Harlan Circuit Court, Harlan, Kentucky 40831, on this 26 day of February, 1976.

  
Of Counsel for Appellees

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### **STATEMENT OF QUESTIONS PRESENTED**

1. Does *KRS 342.316(9)* have to be plead affirmatively?
2. Where all the evidence establishes that myelofibrosis, if not the sole cause of death, substantially contributed thereto, was the Workmen's Compensation Board bound to apportion its award under *KRS 342.316(9)* even if the Board found as a fact that pneumoconiosis "was a probable causative factor" in death?

# SUPREME COURT OF KENTUCKY

File No. 75-1072

---

ROSA BROACH, Individually and as Admin-  
istratrix of the Estate of Sammy S.  
Broach, Deceased, and

WORKMEN'S COMPENSATION BOARD OF KEN-  
TUCKY - - - - - *Appellants*

*v.*

UNITED STATES STEEL CORPORATION;  
JAMES R. YOCOM, Commissioner of Labor  
and Custodian of the Special Fund - *Appellees*

---

APPEAL FROM HARLAN CIRCUIT COURT  
HONORABLE JAMES C. BROCK, JUDGE

---

## BRIEF FOR APPELLEES

---

*May it please the Court:*

Unless otherwise apparent or indicated, throughout this brief numbers in parentheses standing alone refer to pages in the record as indexed by the Harlan Circuit Court Clerk, and emphases in quoted material have been supplied by the writer.

### COUNTERSTATEMENT OF CASE

This is a workmen's compensation case in which the Workmen's Compensation Board found that the employee, Sammy Broach, was totally disabled by pneumoconiosis from January 21, 1970, until his death March 20, 1970, and that pneumoconiosis was a probable causative factor in his death (399-400). Accordingly the Board awarded Rosa Lee Broach as Administratrix of the Estate of Sammy Broach maximum disability benefits from January 21, 1970, to March 20, 1970, and as widow and sole dependent of Sammy Broach maximum death benefits thereafter for a period of 400 weeks from January 21, 1970, or until the termination of dependency (400-401).

Appellees timely appealed to the Harlan Circuit Court (6-8), which rendered an Opinion and Judgment July 16, 1975, remanding the case to the Board with directions that the Board take the following action:

- “(1) Set aside paragraph 2 of its award of March 25, 1974;
- (2) Make findings of fact setting out in percentage terms
  - (a) the extent to which pneumoconiosis contributed to cause death
  - (b) the extent to which myelofibrosis contributed to cause death
- (3) Enter a new decision awarding the plaintiff as compensation that percentage of maximum death benefits that is equal to the percentage to which pneumoconiosis contributed to cause death.” (410-412)

The evidence established that myelofibrosis, if not the sole cause of death, substantially contributed thereto. Thus *Dr. John C. Buchanan*, specialist in internal medicine, treated Broach for some five years before Broach's death (86-89) because of a "malignant disease of the bloodforming tissues, myelofibrosis" (95), "a disease usually affecting the bone marrow primarily" (99) and one "that we consider uniformly fatal eventually" (103). This disease produces an enlargement of the spleen (102) and during the treatment in this case "the focus of interest was on the spleen, which was enormous, and on his blood counts, which brought him here in the first place, and which was causing marked enlargement of the spleen and variation in his blood counts" (92).

During the several years of treatment and in spite of the many chest X-rays routinely done "when a patient comes into the hospital" (91), probably eight or ten series of films in this case (97), Dr. Buchanan "personally did not make a note of a diagnosis of a pulmonary disease" (93) and in the two volumes of records on Broach at Wise Appalachian Regional Hospital Dr. Buchanan did not mention pneumoconiosis until he was contacted by plaintiff's counsel (106-108). *As far as concerns pneumoconiosis, the most that can be fairly concluded from Dr. Buchanan's testimony is that this was a condition that, from the viewpoint of hindsight, did exist but was never considered significant* (93). *Any other interpretation raises the gravest kind of question as to why a known significantly con-*



*tributary disease was never mentioned by the attending specialist.*

Dr. Buchanan's testimony on cross-examination concluded with the following question and answer:

"32. Doctor, let me see if I can make a fair summary statement here. Would you say if I am making a fair statement? As I understand your testimony, this man in your opinion would have died from myelofibrosis at some time and did die from myelofibrosis, but in your opinion the complicated occupational pneumoconiosis contributed to his death to some undetermined extent because in your opinion it hastened his death by some undetermined period of time, whether a day or a week or a month or what you cannot say?

A. I think that is a fair summary of what I said." (109, 110).

*Dr. A. Sidney Barrett* attended Broach during his final hospitalization at University of Virginia Hospital from March 4 to March 20, 1970 (131). The death certificate signed by Dr. Barrett and filed as Exhibit Z of his deposition (136) does not mention pneumoconiosis, silicosis, or occupational disease, and Dr. Barrett candidly admits:

"I don't know whether I'm contradicting myself, *I don't know that I ever said in his chart that he had pneumoconiosis per se.* The X-ray is compatible with it" (141).

While Dr. Barrett concedes that "the exact cause of death is very speculative" (147), *he admits that the drug Busulfan used in the treatment of Broach's*

*myelofibrosis may well have been the immediate cause of death* (152). Specifically he made answer to the following questions:

“Q. In your letter of June 12, 1970, which I will quote now from for the sake of time, you stated that ‘Sam Broach had a peculiar type of chronic pneumonia which has only been recognized in recent years.’ What type of pneumonia is that?

A. I was referring in the letter, although my reference is speculative, although not stated as speculative, I was referring to Busulphan in the lung.

Q. *But he could have died of Busulphan lung, could he not have?*

A. *The immediate cause of death may well have been*” (152).

*Dr. Johnson T. Carpenter, Jr.*, hematologist at University of Virginia Hospital (208), testified as follows:

“Q. Doctor Carpenter, what drug was being used to treat Mr. Broach’s myelofibrosis?

A. I’m going to get the dates here, beginning, I guess when he was here in 1966, a drug called busulfan, B U S U L F A N.

Q. Is that also referred to as myleran?

A. That’s the trade name, yes sir.

Q. Doctor, does busulfan or myleran have any toxic side effects?

A. To get right to the point it does—it is associated with fibrosis in the lung on occasion in some patients.

Q. Does it have other side effects?

A. It has a depressive effect on the bone marrow which is part of why it works.

Q. Dr. Carpenter, does busulfan produce hyperpigmentation?

A. Yes sir.

Q. Mr. Broach have that condition?

A. I believe he did. Let me check the record again. Yes, yes he did.

Q. Does busulfan produce on occasions excessively low white count and platelet count?

A. That's the result of its depressing the bone marrow.

Q. And did that happen in this case?

A. When Mr. Broach came in his white count was at the lower limit of normal and his platelet count was also low.

Q. Doctor, in your opinion, were these conditions and the hyperpigmentation the result of the busulfan therapy?

A. Hyperpigmentation, yes, the low white count and platelet count could certainly have been contributed to by the size of his spleen. We see that quite a bit and he had an enormous spleen.

Q. And what caused the enormous spleen?

A. That's part of the myelofibrosis.

Q. So that whether it was the busulfan or the enlarged spleen, it was still related to the myelofibrosis?

A. Yes.

Q. Now Doctor, getting back to the fibrosis produced by busulfan therapy, I believe that on this man's last admission to this hospital, there was a visible and detectable increase in the fibrosis was there not?

A. Yes sir" (215-217).

\* \* \* \* \*

"Q. March 4, 1970 to March 20, 1970, would you during that period expect to see a change in the type of fibrosis produced by pneumoconiosis or silicosis?

A. No, not in those 16 days.

Q. Yet the fibrosis demonstrated on X-ray examination did increase during this period of time did it not?

A. No, the increasing infiltrate, I'm quoting from March the 12th, has more of an alveolar type of pattern rather than the fibrotic type of pattern. This is a quote from an X-ray report on the 12th, March the 12th. And then on the 15th there was no particular change and then on the 18th there was no particular change, from the last date" (218).

\* \* \* \* \*

"Q. Doctor, do I understand that the, and forgive me if I've covered this, do I understand that the diffuse chronic interstitial pneumonia and fibrosis with giant cell reaction is due to the myelofibrosis or due to the drug used in treatment of that condition?

A. Direct cause and effect is impossible to establish, but this pattern has been seen in individuals who have chronically received busulfan.

Q. In your opinion, within a reasonable degree of medical certainty, was that the cause in this case?

A. Of that pattern I would agree" (221).

\* \* \* \* \*

"Q. Doctor, if I understand your testimony, both the fibrosis produced by pneumoconiosis and the fibrosis probably produced by busulfan, either of these types of fibrosis will cause shortness of breath and respiratory difficulty?

A. Yes sir" (222-223).

\* \* \* \* \*

"Q. Doctor, would this be a fair summary of your opinion, that you feel that the cause of death in this case was respiratory failure due to one, diffuse chronic interstitial pneumonia and fibrosis

with a giant cell reaction which you feel is due to in all probability busulfan therapy and two, nodular fibrosis with doubly refractile crystals, most likely silicosis . . .

A. Yes" (226).

Filed as an exhibit to Dr. Carpenter's testimony was the five-page report from the Department of Pathology (230-234). On the very first page of this pathological report the anatomical diagnosis is set out as follows:

"Treated myelofibrosis with pulmonary fibrosis and gastrointestinal hemorrhage" (230).

Precisely the same language is used in setting out the final diagnosis in the very last sentence on the fifth and final page of the report, which is as follows:

"Final diagnosis: Treated myelofibrosis with pulmonary fibrosis and gastrointestinal hemorrhage" (234).

Again on the first page of the pathological report several accessory conditions are noted (230) which, as set out in the last paragraph of Dr. William H. Anderson's report of April 16, 1973, have nothing to do with the cause of death (269).

The pathology report also notes on the bottom of the second page:

"The radiologists raised the question of possible pulmonary fibrosis as a result of long-term chemotherapeutic drug administration" (231).

On the third page of that report the pathologist states in reference to Broach's condition during those final sixteen days in the hospital:

"The picture of pulmonary fibrosis continued to progress, and the patient required a greater degree of oxygen therapy in order to maintain adequate  $pO_2$ " (232).

On page 4 of the pathology report the cross examination concludes:

"In summary, this is a case of myelofibrosis proved by bone marrow biopsy. The patient then developed fibrosis of the lungs and expired with respiratory failure" (233).

*Dr. William H. Anderson*, after reviewing all the medical reports and depositions three times, prepared a three-page report filed as an exhibit to his testimony (267-269). Dr. Anderson's conclusions in that report are as follows:

- "1) Mr. Broach died at the time he did because he had myelofibrosis that was treated with Busulfan which as one of its known toxic side effects produced pulmonary fibrosis and interstitial pneumonitis with respiratory insufficiency.
- 2) In my opinion he would have died at this same point in time had he not had occupational pneumoconiosis.
- 3) Had he had only occupational pneumoconiosis and had not had myelofibrosis he would not have died at the time he did nor would he have even been disabled for work at that time" (267).

Characteristically Dr. Anderson, who has "had probably more experience than anyone in the country in the study of the effects of pneumoconiosis on arterial blood gases, arterial blood oxygen and gas exchange" (268), supports his position in convincing detail and concludes the third page of his report:

"In summary, I believe the immediate cause of this man's death was pulmonary insufficiency due to Busulfan toxicity in the lungs. This Busulfan reaction in his lungs was probably accelerated by the cessation of Prednisone therapy immediately after his admission to the University of Virginia. 2) In my opinion the pneumoconiosis was not a contributing factor. This opinion is also shared by the pathologist who lists this as only an accessory diagnosis. He also lists as accessory diagnoses chronic cholecystitis with cholelithiasis and hypertrophy of the heart, especially the left ventricle, with scattered myocardial scarring. I agree with this designation of these as accessory and non-contributory conditions. To me it would be just as valid to contend that the scarred hypertrophied myocardium was a contributory cause as to claim that the nodular fibrosis of pneumoconiosis was a contributory cause in his death" (269).

Apparently recognizing the devastating import of Dr. Anderson's testimony, claimant introduced, over the objections of the defense (241, 242, 322), additional evidence in chief under the guise of rebuttal proof in the testimony of *Dr. Richard P. O'Neill*. However, Dr. O'Neill proves to be squarely in agreement with Dr. Anderson when he categorically states:

“Mr. Sammy S. Broach, a 53-year-old white male, died on 3/20/70 of myelofibrosis, treated with Busulfan” (338).

After Dr. O'Neill testified, Dr. Anderson was recalled by the defense and stated in answer to the following questions:

“Q. 21. *Dr. Anderson, Dr. O'Neill states on page 17 of his deposition, 'Mr. Sammy S. Broach, a 53 year old white male, died on 3/20/70, of myelofibrosis, treated with Busulfan.' Do you agree with that statement?*

A. 100%.

Q. 22. Do you find anything in Dr. O'Neill's subsequent testimony that militates against that statement other than his opinion?

A. No” (301-302).

## ARGUMENT

### 1. KRS 342.316(9) Does Not Have To Be Plead Affirmatively.

*KRS 342.316(9)* sets out how compensation is to be apportioned in certain cases. That statute has nothing to do with an affirmative defense.

Indeed two of the cases cited by the plaintiff hold that failure to give due and timely notice is not an affirmative defense that must be specially plead. *Church v. Turner-Elkhorn Coal Company*, Ky., 492 S. W. 2d 877 (1973); *Buckles v. Kroger Grocery and Baking Company*, 280 Ky. 644, 134 S. W. 2d 221 (1939). If failure to comply with the statutory notice requirements is not an affirmative defense, surely it is not



necessary affirmatively to plead *KRS 342.316(9)* in order to insist that the Board follow the Mandate of that statute when the evidence requires that it do so.

2. **Where All the Evidence Establishes That Myelofibrosis, If Not the Sole Cause of Death, Substantially Contributed Thereto, the Workmen's Compensation Board Was Bound to Apportion Its Award Under KRS 342.316(9) Even If the Board Found as a Fact That Pneumoconiosis "Was a Probable Causative Factor" in Death.**

Several evidentiary considerations undergird the conclusion, presupposed in the principle stated immediately hereinabove, that "all the evidence establishes that myelofibrosis, if not the sole cause of death, substantially contributed thereto."

1. Neither the specialist who had Broach under his care for a period of five years before death, Dr. Buchanan, nor the physician who had Broach under his care during the final sixteen days of hospitalization, Dr. Barrett, ever indicated to anyone that the deceased had pneumoconiosis prior to death. This point is emphasized here and hereinabove not for the purpose of contending that Broach did not have pneumoconiosis—he most certainly did have—but rather for the purpose of underscoring the fact that pneumoconiosis was never considered to be a significant problem in this man's case.

2. Dr. Buchanan unequivocally testified that myelofibrosis is inevitably fatal, that Broach would have died, and in fact did die, from myelofibrosis, but that pneumoconiosis contributed to his death to some un-

determined extent because it hastened death, although he would not say whether it hastened death by a day or a week or a month or whatever. Clearly this is a theoretical, nebulous expression of opinion that leaves the whole matter in the realm of speculation or, at best, suggests that the contribution of pneumoconiosis to death was *de minimis*.

3. Dr. Barrett flatly admitted that the drug Busulfan "may well have been" the immediate cause of death.

4. The pathological report filed with the deposition of Dr. Johnson T. Carpenter, Jr., does not implicate silicosis or pneumoconiosis as a causal factor in death. The changes of occupational disease are listed only in the list of non-contributory "accessory" diagnoses, and the anatomical or final diagnosis as to cause of death is "Treated myelofibrosis with pulmonary fibrosis and gastrointestinal hemorrhage." All the proof shows that myelofibrosis causes pulmonary fibrosis, and it is clear that the "pulmonary fibrosis" mentioned in the anatomical or final diagnosis is the pulmonary fibrosis that accompanies myelofibrosis. Again, the nodular fibrosis of occupational pneumoconiosis is mentioned only in the list of accessory diagnoses.

5. Claimant's Dr. O'Neill made the categorical statement with which the defense agrees:

"Mr. Sammy S. Broach, a 53-year-old white male, died on 3/20/70 of myelofibrosis, treated with Busulfan" (338).

6. Dr. O'Neill makes two similar statements that are highly significant:

"The basic pathological lesion seen in Busulfan lung is that of an alveolitis and to a lesser degree, true interstitial fibrosis" (339).

\* \* \* \* \*

"Busulfan lung causes classically an alveolar fibrosis and to a very much lesser extent interstitial fibrosis" (343).

Thus Dr. O'Neill emphasizes that the pulmonary fibrosis usually seen with Busulfan lung is of the alveolar type, and this is precisely the type of fibrosis that Dr. Carpenter said progressed so rapidly during the last sixteen days of this man's hospitalization. Again quoting Dr. Carpenter:

"Q. March 4, 1970 to March 20, 1970, would you during that period expect to see a change in the type of fibrosis produced by pneumoconiosis or silicosis?

A. No, not in those 16 days.

Q. Yet the fibrosis demonstrated on X-ray examination did increase during this period of time did it not?

A. No, the increasing infiltrate, I'm quoting from March the 12th, has more of an alveolar type of pattern rather than the fibrotic type of pattern. This is a quote from an X-ray report on the 12th, March the 12th. And then on the 15th there was no particular change and then on the 18th there was no particular change, from the last date" (218).

Perhaps no other testimony in this case is more significant than that referred to immediately hereinabove. One of the major issues in this case is with

reference to the kind of fibrosis that was progressing so rapidly during the final days of this man's life. Dr. O'Neill says that alveolar fibrosis is classically caused by Busulfan and Dr. Carpenter says that it was the alveolar pattern that was increasing so rapidly during Broach's final days.

7. No testimony actually refutes the well-reasoned definitive opinion of Dr. William H. Anderson and, as a matter of fact, close analysis of all of the actual facts in the case, as distinguished from unfounded opinions, undergirds Dr. Anderson's conclusion that pneumoconiosis had nothing to do with the death of Samuel Salvadore Broach.

Before the Board claimant pitched her case squarely on *Young v. Holbrook*, Ky., 472 S. W. 2d 473 (1971), and the Board's citation of that case in its Rulings of Law coupled with its language about "probable causative factor" in Finding of Fact No. 7 (400) makes clear that the Board bought the claimant's argument. However, *Young v. Holbrook* was a proceeding under *KRS 342.111* by the widow of a deceased employee to recover the balance of compensation awarded her husband who died before the full award had been paid.

Likewise *Falcon Coal Co. v. Sweet*, Ky., 518 S. W. 2d 343 (1974), cited by claimant on this appeal, was a proceeding under *KRS 342.111*. *The instant case is not a proceeding under that statute*. Indeed it could not have been since *KRS 342.111* was not even effective when decedent last worked. *Trimble v. United Fuel Gas Company*, Ky., 481 S. W. 2d 658 (1972).

Accordingly *KRS 342.316(9)* is pertinent:

“Where an occupational disease is aggravated by other disease or infirmity not itself compensable, or where disability or death from any other cause not itself compensable, is aggravated, prolonged, accelerated or in anywise contributed to by occupational disease the compensation payable shall be reduced and limited to such proportion only of the compensation that would be payable if occupational disease were the sole cause of the disability or death as such occupational disease, as a causative factor, bears to all causes of such disability or death. . . .”

*This statute simply cannot be ignored in this case where all the proof establishes that myelofibrosis was either the sole cause of death or at least substantially contributed thereto. Stated differently, if the statute is not applicable to this case, to what kind of case would it be applicable?*

In passing it should be noted that claimant completely misreads *Pond Creek Colliery v. Taylor*, Ky., 302 S. W. 2d 838 (1957). There the Court held that the claim came under the silicosis-tuberculosis statute, *KRS 342.316(7)*, rather than under *KRS 342.316(11)*, which was *KRS 342.316(9)* at the time Broach last worked. Certainly Broach's case does not fall under the special silicosis-tuberculosis statute.

**CONCLUSION**

If *KRS 342.316(9)* has any meaning at all, and unless it is to be repealed by judicial fiat, it is believed that this Court will have no alternative but to affirm the Judgment of the Harlan Circuit Court.

Respectfully submitted,

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